[Int. Arch. Allergy Immunol., **107**, 587-591 (1995)] [Lab. of Pharmacology]

Relationship between histamine release and leukotrienes production from human basophils derived from atopic dermatitis donors.

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We examined the relationship between histamine and leukotrienes (LTs) production from basophils in the presence or absence of 1 ng/ml of interleukin-3 (IL-3). Normal basophils released a smaller amount of histamine and LTs than atopic dermatitis (AD) basophils stimulated with an optimal concentration of anti-IgE antibody. When we examined the relationship between these mediator release from AD donors, we found a significant exponential correlation between these two mediators. Although IL-3 enhanced both histamine release and LTs production from AD donors, the relationship between these two mediators was not affected.

[Pharmacol. Commun., **6**, 361-367 (1995)] [Lab. of Pharmacology]

Inhibitory mechanisms of prednisolone for delayed type hypersensitivity reactions in mice.

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The inhibitory mechanisms of prednisolone (Pred) on the effector phase of delayed type hypersensitivity (DTH) reactions caused by picryl chloride and tuberculin were investigated in mice. Pred administered intraperitoneally 6-22h after challenge significantly inhibited both DTH reactions. The inhibition caused by Pred given 14h after challenge was not affected by 17 α -methyltestosterone or cycloheximide. On the contrary, the agents completely abrogated the inhibition caused by Pred given 22h after challenge.

[Int. Arch Allergy Immunol. **108**, 189-195 (1995)] [Lab. of Pharmacology]

Effects of some immunosuppressors on allergic bronchial inflammation and airway hyperresponsiveness in mice.

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The effects of two new immunosuppressors, FK-506 (FK) and mizoribine (MZ), on antigen-induced bronchial inflammation and reactivity to acetylcholine in mice were studied in comparison with those of cyclosporin A (CsA) and cyclophosphamide (CP). The administration of each of the four immunosuppressors clearly inhibited the airway eosinophilia. Moreover, FK, MZ and CP clearly inhibited the interleukin-5 (IL-5) production and CsA showed the tendency to inhibit IL-5 production. Whereas FK, MZ and CsA clearly inhibited the airway hyperreactivity, CP did not show a significant effect on this airway hyperreactivity. These two drugs inhibited the airway hyperreactivity, probably by another mechanism to inhibit airway eosinophilia and IL-5 production.